exclusion chromatography (SEC). Interaction studies with the proteins and the specific mRNA and/or DNA *in vitro* for example via electrophoretic mobility shift analysis will follow.

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7P10

A mitochondrial reporter system for studies on NRF-1/AMPK activity and regulation in viable cells

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Mitochondrial biogenesis is subjected to complex physiological control and mitochondrial mass and capacity vary according to the cell specific demands for respiratory energy.

Abundance of mitochondria can be modulated in response to physiological conditions such as exercise, cold exposure, caloric restriction and oxidative stress, cell division and renewal, and differentiation

We have generated a mitochondrial targeted GFP reporter cell model system to be able to study mitochondrial biogenesis. The cells are stably expressing a reporter construct regulated by nuclear respiratory factor 1, NRF-1. This transcription factor is essential for transcription of genes contributing to the mitochondrial DNA transcription machinery and genes encoding components of the respiratory chain among others.

Containing a NRF-1 regulated and mitochondria targeted GFP construct, the cell model simultaneously reports transcriptional regulation of NRF-1 target genes, as well as parameters of mitochondrial mass and morphology.

The AMPK activator, AICAR, has been used to induce mitochondrial biogenesis in these cells and various molecular techniques have been used to study the effects of NRF-1 activation on mitochondrial biogenesis. Repeated stimulation and cell-sorting were employed to select populations presenting dynamic regulation of mitochondrial biogenesis.

Different known activators of mitochondrial biogenesis are currently being used to test the reporter system in both malignant and non-malignant cell lines.

This model system provides unique possibilities to identify new mechanisms regulating mitochondrial function.

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7P11

Cytochrome *c* oxidase assembly factor Surf1: Candidate for heme *a* insertion into subunit I

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Biogenesis of the mitochondrial cytochrome *c* oxidase (COX) is a highly complex process involving more than 30 known accessory proteins. Here we focus on the steps taken by heme *a* from its site of synthesis, heme *a* synthase (HAS), to its final target site in COX subunit I, using the soil bacterium *Paracoccus denitrificans* as model organism. Studies in humans have shown that the dysfunction of the Surf1 protein is associated with Leigh syndrome, exemplifying the crucial role of this protein in cytochrome *c* oxidase maturation. Its heme binding properties, which have been confirmed *via* isothermal titration calorimetry for both *Paracoccus* homologues (Surf1c and Surf1q) [1] imply a role as heme stuttle. Further *in vitro* and *in vivo* interaction studies between Surf1 and heme *a* synthase support the idea that Surf1 is responsible for the specific uptake of heme *a* from HAS, its sequestration and coordinated insertion into COX subunit I [2]. Our current focus is now on the detailed elucidation of the molecular interaction of heme *a* with Surf1 on the one hand and HAS/Surf1 on the other hand, employing X-ray crystallography and solid state NMR techniques.

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7P12

Mgr2 promotes coupling of the mitochondrial presequence translocase to partner complexes

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As most mitochondrial proteins are encoded in the nucleus and synthesized as precursors in the cytosol, they must be post-translationally imported into mitochondria. After passage of the outer mitochondrial membrane via the TOM complex (Translocase of the Outer Membrane), precursor proteins with N-terminal, cleavable presequences are passed on to the TIM23 complex (Translocase of Inner Membrane 23), which mediates their further sub-mitochondrial sorting: Percursors with an internal hydrophobic stop-transfer signal are laterally released into the inner membrane, whereas water-soluble precursors without such membrane-insertion-signals are translocated to the mitochondrial matrix with the help of an ATP-driven import motor.

We have employed a systematic proteomic approach to analyse the composition of the TIM23 complex and identified the inner membrane protein Mgr2 as a novel genuine subunit of this translocase. Mgr2 recruits the regulatory component Tim21 to the essential TIM23 core complex. The Mgr2/Tim21 module is required for the efficient coupling of respiratory chain supercomplexes to the TIM23 machinery. Association of TIM23 with the respiratory chain facilitates the membrane-potential-dependent step of precursor protein insertion into the inner mitochondrial membrane. Moreover, Mgr2 plays an important role in the hand-over of precursor proteins from the TOM to the TIM23 complex. Consequently, Mgr2-deficient